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VOL. XV. NO. 4. OCTOBER 20TH, 1921.

STORAGE

TRANSACTIONS

OF

THE ROYAL SOCIETY OF TROPICAL MEDICINE AND HYGIENE

London:
ROYAL SOCIETY OF TROPICAL MEDICINE AND HYGIENE,
11, CHANDOS STREET, CAVENDISH SQUARE, W. 1.

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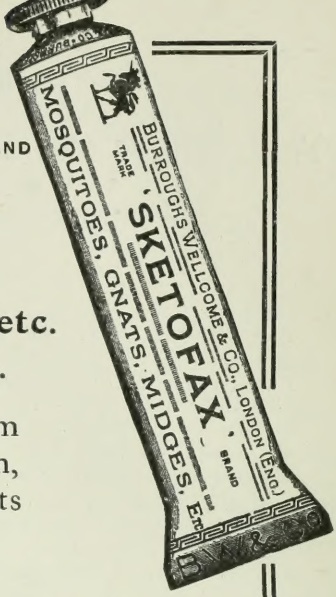

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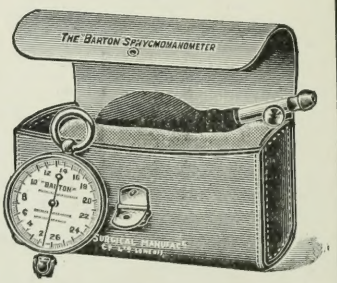

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TRANSACTIONS

OF THE

ROYAL SOCIETY OF TROPICAL MEDICINE AND HYGIENE.

OCTOBER 20TH, 1921.

VOLUME XV. No. 4.

Proceedings of a meeting of the Society held on Thursday, October 20th, at 8.15 p.m., at 11, Chandos Street, Cavendish Square, W. 1, Sir JAMES CANTLIE, K.B.E., F.R.C.S. *(President)*, in the Chair.

LIFE INSURANCE IN THE TROPICS.

Inaugural Address.

BY THE PRESIDENT, SIR JAMES CANTLIE, K.B.E., F.R.C.S.

I am very much obliged to you for the kindly reception you gave me the other night, at my installation as President, and again to-night.

The subject I have chosen for what is called my Presidential Address is one which I think interests every one in the room; you are every one interested in the tropics, whether medical man or layman. It is an absorbing question. Amongst us are medical officers of insurance companies, medical examiners for companies, banks and commercial firms, for members of their staff who are about to proceed to the tropics, and who examine their men on their return on leave.

I have interested myself in the matter for some twenty years, and published several articles and read several papers on the subject. As far back as 1897, a paper was published in the *British Medical Journal*; I addressed a meeting of the Life Assurance Medical Officers'

Association in 1903 ; and, further, read a paper before the Royal Institute of Public Health at one of their Conferences.

The question before us seems to resolve itself into—Does residence in the tropics impair the health of the individual to such an extent as to justify the imposition of an extra premium being demanded by insurance companies. In insurance prospectuses I see that a dangerously warm country is regarded as one situated within 33° north of the equator and 30° south of the equator.

It is, of course, well known that within the area specified we meet with climates of widely different characteristics, varying between climates of an equatorial, a tropical, a sub-tropical and a temperate character ; climates, some with an insular and some with a continental type of character. To slump these together, as some of the companies say they do, is perhaps an inevitable method of dealing with such a huge subject as tropical insurance, but it is hard on individuals proceeding to a " good climate " area within the specified longitude and latitude, and one wonders how the rules appertaining to the amount of extra premium demanded were drawn up. Was it from statistics of the percentages of deaths published by the local governments in Crown Colonies, etc., or was it from information received from travellers ? If not, one cannot conceive what were the sources from which the knowledge was derived—say fifty years ago. I have described the tales we hear of these distant tropical lands as ship captains' gossip gathered from the common talk of those lands when the ship goes in harbour. On the way out in 1887, the officers of the ship—whether sailors or engineers—spoke of little else than the fate of the passengers whom they had taken out on previous voyages, how they were dead in twelve months or sent home wrecks after a few years. When again one's friends and relations at home heard one was to go to the eastern tropics, one was deluged with letters, cuttings from reports, callers beseeching one to give up the idea of going out to a death-trap, a " white man's grave," and so forth, until one wondered how any Briton ever went out at all.

No wonder some of the Insurance Companies of that date said they would have nothing to do with insuring the lives of persons going to the tropics on any consideration whatever. Were the extra premium charges founded on the local government reports, here, again, the information was faulty in a superlative degree. None of our tropical colonies

produces its own labour; the white population is imported from Britain and the native labour from the neighbouring countries, or brought from overseas: Tamils, etc., to Ceylon, Chinese to the Malay States and to the Straits Settlements, Indians and Chinese to the West Indies, and so forth. They come as young adults as a rule without their wives and children; without, in fact, the part of the family which swell the death-rates in home countries. The white population consisted of bachelors for the most part, who, on being carefully examined, were sent out in perfect health; often taught by lectures how to avoid diseases; re-vaccinated against small-pox, inoculated against typhoid, and occupying dwellings—often of a better class than they were accustomed to at home.

When they get ill they are carefully attended to, sent for a change or sent home on sick leave. What are the diseases that give the district a bad name? Malaria is the chief, but malaria kills but a small percentage of those attacked. It is a disease that incapacitates, and the loser is the employer or the firm in which they work—not the insurance office, whether the insurance is for a period of years or for life.

Besides, malaria, its cause, prevention and treatment is so well known at the present day that its danger is fractional.

Compared to that of pre-LAVERAN-MANSON-ROSS days, it may be said that if a man gets malaria in modern times the fault is with the man, because he did not follow the precautions now so well known.

No insurance company, I take it, would have insured the lives of Drs. LOW and SAMBON when they went to live in the Roman Campagna during the most malarial periods of the year, yet they came back without being infected and in excellent health. The mosquito-net was the sole protector. So it will be in all malarial lands. Anyone can defy malaria who wards off the mosquito. And not only malaria, for the U.S. Army Commission has proved that yellow fever can be prevented in a similar manner. In the West Indies, therefore, where malaria and yellow fever are the scourges, there is no reason why one should contract either of these diseases. So keenly has this come home to us, that many declare that the tropics are dangerous to the white man only through their diseases, not through their climate. Ward off malaria, and health is capable of being enjoyed in any climate. Whilst not, perhaps, going so far as to say that climate has no deleterious effect, I do claim that in

the tropics, if malaria can be prevented, the white man can resist the effect of a hot climate for an indefinite time.

In temperate climates we have to build our homes so as to keep out cold, damp, and thereby protect us from lung troubles, so the tropical resident has to build his house a "protected house" to keep out the mosquito.

Beyond malaria, which involves the insurance companies to but an infinitesimal extent nowadays, the other disease of importance is dysentery and its sequelæ. That is, again, greatly lessened to-day by the emetine treatment of ROGERS. We are quite aware that emetine leaves a tendency to post-dysenteric colitis, perhaps to a greater extent than the ipecacuanha treatment of pre-emetine times, but the death-rate from acute amœbic dysentery has been lessened to a marvellous extent, and although colitis is perhaps more prevalent as a sequela, the man is sent home as an invalid, and the tropical region where he contracted the disease is freed in its statistical records from the old-time death-rate from this disease.

What, then, is an extra premium charged for, as other diseases are but on a platform with home ailments? These facts I have brought forward on several occasions, and whether in consequence of my speaking and writing I know not, but several companies now advertise world-wide policies. In a short time all companies must follow suit, otherwise the world-wide policies will kill out the still perverse companies.

The great diseases of the tropics affect, for the most part, the young men—the unacclimatised, as they are called. It is during the first two or three years of tropical life for young men going out that the tropics is dangerous, and the younger they go out the greater the danger. Time was when men went out at a younger age than now. Sixteen or seventeen was a common age to send out young men some thirty years ago. Gradually the advantages of sending young men out at about twenty became apparent, and for a man to go out before twenty-one is looked upon to-day as inadvisable and dangerous. Twenty-three is the better age, and twenty-five the best. This fact, that the earlier the age the more the danger, affects insurance. Before going out the young man is likely to be insured by his parents or guardians; and as the younger the age the better the terms of the policy for home life, he may

be insured, say, in the office at home when seventeen When, however, he goes overseas to the tropics, the extra-premium question arises. Abroad, his salary barely keeps him, and he has £10 or £20, or in some cases even £40, charged him, over and above the home premium. He is now free from parental help and control, and the extra charge falling upon him either runs him into debt or he lets the policy lapse, and the money he paid is lost to him and given to the benefit of the company, "for the risk they have run."

I have brought forward more than one plan whereby this dropping of policies may be prevented. Of these attempts to lessen the chance of the young man allowing the policy to lapse, I suggest the following scheme as one which would accomplish this:—

Supposing a young lad of seventeen is insured, whilst working in an insurance office at home, for £1,000. When he is twenty years of age he is sent overseas in the company's service. Up to then his father paid his insurance, but when he goes abroad an extra premium is imposed and his father expects him to pay the premium on his life. He finds after a year or two the premium, plus the extra premium imposed, is more than he can meet and he allows it to lapse.

To avoid this occurring, I would suggest that instead of the company charging the extra premium, that the company reduce their liability, and that, instead of being liable for the £1,000 originally taken out, the liability of the company be reduced to, say, £500 in case of the death of their client, or it might be to £750 if the climate is a "good" one. This should hold good for say five years, that is to say, until the insured has passed through the "dangerous period" of his tropical residence. If at the end of the five years, and if the client can produce a doctor's certificate to the effect that he is quite well I suggest that the extra premium be abolished altogether, or at least reduced by half, and their full liability of £1,000 be restored. Instead of these hard and fast lines being followed, the restoration might be graded. If it is reduced, to begin with, by £500, after a period the liability might be raised to £700, and, after a few more years be graded up to £1,000.

The authorities of the insurance office scout this proposal as they scout any proposal made by outsiders, and especially by medical men. The actuaries are the sole authorities, and their laws and opinions are those of the Medes and Persians.

I have been told by members of insurance companies whom I invited in 1908 to an address I was giving, that it did not matter what I said, however good it might be, that I was fighting a "close" body; that by to-morrow noon, the messengers from the various offices would meet each other and agree whether the office were to take any notice of the scheme or not—of course, they were not.

This, however, did not stop me, and I have lived long enough to see several of the measures repudiated by them at the time, now in full play. The change was an actuarial one, and not as a consequence of interfering doctors and other men ignorant of the calculations of the mathematically evolved schemes necessary to regulate insurance. The doctors have the practical side of the consequence of insurance before them and do not need mathematics to help them to conclusions. The doctors are the sole ultimate authorities, and no juggling with figures can alter their opinions.

When I read my paper before the Life Insurance Medical Officers' Association in 1903, Sir DYCE DUCKWORTH was in the chair, and he, in commenting upon my paper, said, "Mr. CANTLIE'S plan of dealing with extra premiums for young men would free the young man from increasing his payment, and at the same time diminish the risk of the company satisfactorily. I do not know if business men would accept the principle, but to me it seems feasible and fair. I happened to see the manager of my own insurance office to-day, and I put this to him. He said it was nonsense on the face of it, and that no insurance office would contemplate such a matter as sound business. He went on further to repudiate several of the assertions of Mr. CANTLIE throughout this paper, which he had read, and to say that these matters do not, as Mr. CANTLIE says, rest entirely on the *dicta* of the doctors." He says, "I would recommend British insurance offices to rely for advice as regards the value of the lives of tropical residents on the medical officers of their companies, and not on their actuaries. The actuary cannot know, he has no means of knowing, the effect of climate or the local state of healthfulness in any individual colony." Then he goes on to say that "the actuary's figures are misleading, and his statistics of no use to him there. To-day I was told that that was not the case. I was told there was a great deal of mathematics in these cases, and that it is not merely an opinion formed on mere climate which the person goes to,

but there are actuarial matters of great importance which we doctors are not supposed thoroughly to understand." Something wants doctoring, and it is not the doctor, but—

The Insurance of Natives.—The question of insuring the lives of natives remains a vexed one. British offices, for the most part, do not push this business; American insurance offices do. I am not prepared to give you a statistical table of the expectancy of life of, say, a Hindoo, Malay, Chinaman, or negro: no statistics are available; but I am in a position to express an opinion in regard to the matter. The question narrows itself down to a very fine point. It is only the more advanced and intelligent of the natives of any Asiatic country who take out insurances on their lives. It is the natives who have become "Anglicised" to a greater or less extent that usually come forward for insurances. But in the process of becoming Anglicised they have acquired more than an imperfect knowledge of English and British habits in business. They affect British ways of living to some extent, and especially do they take to the food and drink which they see supplied to the British table. Beef or mutton take the place of fish or fat pork in the diet, and European wines, especially champagne, are the constant concomitants of, say, the Chinaman who takes to European "chow." A rich Chinaman who affects the European mode of living has his Chinese "chow" with his family, and European "chow" (it may be immediately afterwards) with his Anglicised *confrères*. The excess of rice at one meal, with beef, etc., and sweet champagne—the sweeter the better he likes it—in time tells its tale. Gout, kidney and liver troubles supervene, and his days are shortened. It is from this very class that most native lives are insured, and they are the very worst lives of the land they belong to. The ignorant and simple living native might be as good a life as any European—as a matter of fact he seldom is—but when he departs from his simple habits, and takes to "foreign" ways of eating and drinking, his life is not a good one. I saw much of American methods of dealing with the Chinese in regard to insurance. At one time, in Hong Kong, the Chinese insured largely in American insurance offices, but during the last years of my stay there they almost ceased doing so. Perhaps they have taken it up again, but I hope on a different footing to that which was in vogue when I knew the methods followed.

Insurance Anomalies.—The insurance offices say, "Well, we slump the risks, and what we lose by the younger men we gain on those of more mature years." Many hardships are, however, incurred therefrom. But yesterday I saw a nurse proceeding to one of our colonies, a healthy woman of forty, who had to pay an extra premium of £10 a year; her actual annual salary amounting to about £60. I know also of *patients*, who, on being recommended to go to a warm climate by their physician, had to pay an extra premium to their insurance offices—a most anomalous state of affairs. They were being sent to the only place in the world where health could be obtained and where their lives could be prolonged, yet they are asked to pay an extra premium for their doing so.

I have known men who, after long residence in a warm climate, find they cannot keep their health at home, and go abroad again to live. They have paid an extra premium on their life assurance whilst abroad; this has been rebated when they came home, but again enforced when, in order to live at all, they have gone to the part of the world which suits them best; the insurance office in this instance remitting the extra premium in the only place where their client could not live, namely, at home.

Discussion.

Sir William Leishman expressed the pleasure with which he had listened to the President's excellent address, which was full of common sense. It was up to the tropical members of the profession to put the insurance companies right. Actuaries took the line they did presumably because of defective information. Sir James said there were no proper statistics extant, but the speaker was doubtful as to that. He believed careful delving among the records of some of the Crown Colonies would produce such statistical evidence as might persuade the actuaries that the supposed danger to young life in the tropics was greatly exaggerated. It was very much to the detriment of the Empire that such an impression should be widespread. If the words of Sir James Cantlie should reach and impress the insurance companies concerned, they would have done much good.

Sir Leonard Rogers said the President had raised one or two points of practical importance. One was as to whether the Society should form a Committee which could give information relative to insurance. A very important factor was the enormously improved prospects of healthy life in the tropics now as compared with not very many years ago, owing to the great advance in tropical medicine; for instance, inoculation against typhoid. In Calcutta he found that 80 per cent. of the attacks of typhoid in the General Hospital, among Europeans, were in the first three years of their residence there. That had been the greatest cause of death in India and other parts of the tropics, and was now easily preventable. The same could be said of cholera, dysentery and liver abscess; and if the insurance companies would take cognisance of these facts and reduce their rates, it would be a matter of great practical importance.

Another point was as to Americans being so far more enterprising than English companies. Some years ago he spent much time in analysing 4,000 post-mortem records in Calcutta, working out the causes of death and the implication of various organs, and he never once got a reference to that work from any English insurance company's officer; but he had heard from one of the biggest American companies that it had reprinted the whole report of the investigation for the use of their officers in America.

The following gentlemen also took part in the discussion :—Lieut.-Col. Clayton Lane, Lieut.-Col. J. C. Kennedy, Squadron-Leader H. E. Whittingham, Drs. Leopold Hill and A. T. Ozzard, and Messrs. H. E. Hornby and A. L. Sheather, and the President replied.

SHORT PAPERS.*

NOTE ON RELATION BETWEEN TSETSE-FLY (*GLOSSINA MORSITANS*) AND GAME IN THE PROCLAIMED AREA, NYASALAND.

By WILLIAM A. MURRAY, M.B., Ch.B.

The Glossinæ are fairly widely distributed over British Nyasaland, but are chiefly found in the narrow forest belt which covers the west shores of Lake Nyasa, from Chintechi in the north to Fort Johnson at the south end, then extends along both banks of the Upper Shiré as far as the Murchison Cataracts opposite Blantyre (see map).

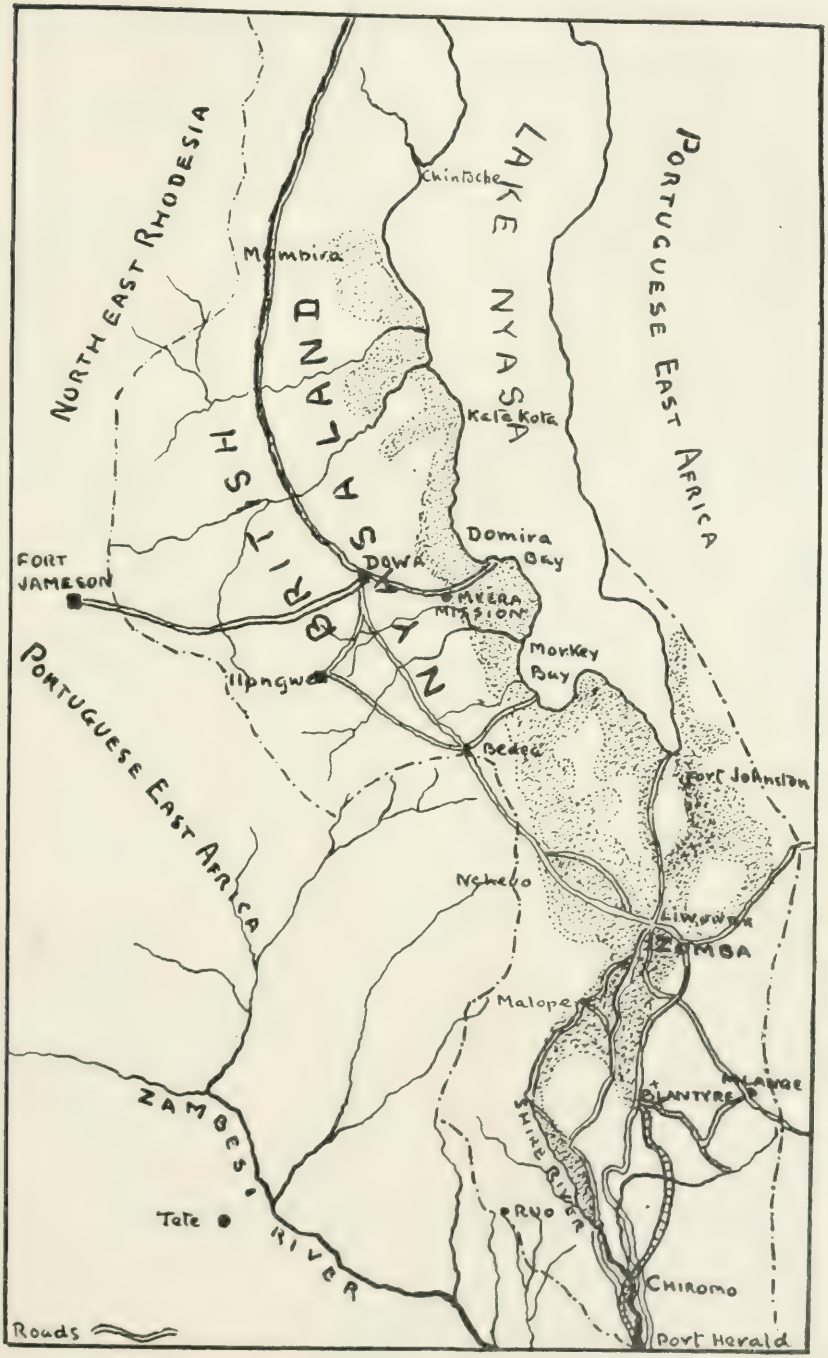
Of these, *Glossina morsitans* is by far the most important and the most widely distributed species. *Brevipalpis, fusca* and *pallidipes* are also found, but more rarely.

During the writer's residence of twelve years at Mvera, Central Angoniland, on the border of this fly-infested forest, he had an opportunity of carefully observing the fly and its movements in the section of forest adjacent, extending roughly for fifty miles from north to south and twenty-five miles in width.

When the writer first arrived at Mvera Station (which is situated just outside the western border of the forest, and is about twenty-five miles west of Domira Bay) he found the whole adjacent section of forest (which was afterwards proclaimed as the Quarantine Area for Sleeping Sickness) quite clear of tsetse-fly, except at one spot near the south-east corner of this area. At the same time there was very little game to be found in this forest, except at the same place.

Enquiries from old residents at Mvera elicited the fact that prior to 1896, when the rinderpest swept through the land, and decimated alike native cattle and all kinds of large game, the latter had roamed freely over practically the whole of the forest, and that wherever game was found there was also tsetse-fly. This information was confirmed by

*With a view to making the Transactions more interesting, it is proposed to insert SHORT COMMUNICATIONS from Fellows relative to any matters of medical, sanitary or parasitological interest. It is felt that many Fellows in various parts of the world must be in a position to contribute short papers on these subjects, and the Secretaries will be pleased to receive any such from them.



Map shewing TSETSE FLY AREAS in NYASALAND

Sir ALFRED SHARPE, then Governor of the Nyasaland Protectorate, during a conversation with the writer at Dowa, in 1902.

Sir ALFRED had been a mighty hunter in the early years of the Protectorate, and well remembered that part of the forest for the abundance of both large game and tsetse-fly.

It was clear, therefore, that the rinderpest had not only almost wiped out the game, but that the tsetse-fly had also diminished to a corresponding extent all over that large area. It would be of great interest if one could find out whether the fly had died as a result of imbibing the blood of sick and dead animals, or whether they died off, or departed elsewhere, because of the subsequent great scarcity of mammalian blood as food. It may be said that, prior to 1900, the whole forest was very sparsely inhabited by the Achewa natives of the western plateau, owing to their dread of their hereditary enemies the slave-hunting Yaos, who live all along the lake shore.

The small herds of buffalo, eland, kudu, hartebeest and sable that had survived the rinderpest mostly took refuge in the Tsanjoka Marsh, some ten miles south of Domira Bay, and for some years until about 1904, practically confined themselves to that locality. Buffalo, eland and sable were also found at the Lingadzi River, near the northern boundary of this area, but there were no tsetse whatever at that time at the Lingadzi. As, however, game increased in numbers, and as also natives began to establish villages near both these localities, the game commenced to scatter over a wider area of forest, and gradually returned to their old haunts over the greater part of the forest.

The fly which had been strictly limited for six or eight years to the Tsanjoka Marsh, spread concurrently with the game. This was of great concern to the Europeans at Mvera, whose cattle were accustomed to graze down in the forest, and who depended mostly on donkeys for transport while travelling about in the forest. They found from 1904 onwards that the tsetse-fly free area became gradually more and more restricted, until by 1908 the fly was seen within a mile of Mvera Station. This experience was confirmed by the loss of several donkeys and cattle from nagana disease (the type was *T. pecorum* in all cases examined by the writer).

The foregoing account of the diminution and spread of tsetse-fly over a comparatively small section of the lake shore forest serves as an

example of the close connection between the movements of game and tsetse-fly over the whole Nyasaland fly area. This, of course, only applies to the low-lying parts of that forest, as beyond an altitude of 3,000 feet tsetse-fly was rarely found in Nyasaland, even though game was abundant in otherwise suitable country.

The writer's experience, therefore, fully corroborates the paper by Mr. W. KIRBY GREEN, published in the *Minutes of Evidence* taken by the Parliamentary Commission on Sleeping Sickness, in June, 1914, p. 253.

HISTORY OF THE INTRODUCTION AND SPREAD OF HUMAN TRYPANOSOMIASIS (SLEEPING SICKNESS) IN BRITISH NYASALAND IN 1908 AND FOLLOWING YEARS.

BY

WILLIAM A. MURRAY, M.B., CH.B.

Late Captain S.A.M.C. and Officer-in-Charge Sleeping Sickness Investigation in Zululand.

INTRODUCTION.

The writer arrived in Nyasaland in March, 1901, and was in charge of a Native Mission Hospital at Mvera, about twenty-five miles west of Domira Bay, on the west shore of Lake Nyasa, until December, 1912, and had unusual facilities for observing the incidence and spread of human sleeping sickness in Nyasaland. Not only were all the first cases associated with his district, but he was asked by the Nyasaland Government to take charge of the Sleeping Sickness Isolation Camp for eight months during 1910 and 1911, and in 1912 was permitted, through the kindness of Colonel (now Surgeon-General) Sir DAVID BRUCE, C.B., to associate himself with the investigations carried out by the Sleeping Sickness Commission at Kaso, near Mvera. The results of their labours were published in 1914 as the *Sixteenth Report of the Sleeping Sickness Commission of the Royal Society*, and to this valuable report I am indebted for many of the following facts and references. For other data I am indebted to the official *Nyasaland Sleeping Sickness Diaries*

(Nos. 1 to XXV., published between November, 1907, and December, 1915, and then discontinued).

WAS SLEEPING SICKNESS ENDEMIC IN NYASALAND OR INTRODUCED?

The Royal Society's Commission came to the conclusion that "Nyasaland Sleeping Sickness has not been introduced in to Nyasaland from Lake Tanganyika, but is an endemic disease of the country," basing their opinion on "papers by Drs. SANDERSON, SHIRCORE, and others." From this opinion I dissented strongly at the time, as Colonel BRUCE admits in his Report quoted above (*vide* pp. 34 to 38 for the argument *in toto*), and I have seen no reason since then to alter my conviction.

The following account of the manner in which the disease broke out in Nyasaland is part of the proof of my opinion that there was no sleeping sickness in Nyasaland prior to 1907 or 1908, and that it was introduced about that time from Lake Tanganyika or North-Eastern Rhodesia, or both.

HISTORICAL SURVEY OF SLEEPING SICKNESS.

Prior to 1884, human sleeping sickness (due to a protozoal blood parasite, *Trypanosoma gambiense*, and spread by an intermediary host, *Glossina palpalis*) was confined to the West Coast of Africa and the region of the Lower Congo. After the war against the Arab power in the Congo, in 1892, the disease spread widely over the Congo basin, due to large military movements. In 1896 the disease appeared in Uganda following the arrival of EMIN PASHA'S followers from the Congo Free State, who settled in Uganda. The disease spread rapidly about the shores of Lake Victoria Nyanza, and claimed about half a million victims between 1896 and 1904. By 1900 the disease had extended to Kasongo, south-west of Lake Victoria Nyanza, then spread to Lake Tanganyika and the Lualaba River towards the Katanga district.

At the end of 1907 the same disease (still caused by *T. gambiense* and carried by *G. palpalis*) had spread as far south as the southern shores of Tanganyika and Lake Mweru. By July, 1908, cases had been found along the Luapula River in North-East Rhodesia and near Lake Bangweolo. Investigation during 1907 and 1908 in North Nyasa districts proved that men of the Atonga tribes had been travelling in

hundreds to and from Katanga and other infected districts of Tanganyika in search of work, so that there was every likelihood of infection being carried back by them to their homes in Nyasaland.

In 1908 cases of a more virulent type of sleeping sickness (later known as the Rhodesian type) were found in North-Eastern Rhodesia, near the Nyasaland border, and investigation showed that infection had taken place in or near the Luangwa Valley where no *G. palpalis* existed, but only *G. morsitans*, which up to that time had not been seriously considered a potential carrier of sleeping sickness.

The Nyasaland Government had meanwhile not been idle, but had closed the Rhodesian-Nyasaland Border in 1907 to all native travellers except at a few posts, where medical observation of all travelling natives was to be carried out, in order to prevent if possible the entry of infected natives into Nyasaland This measure proved futile, as the natives easily evaded the posts and the police.

Further, the Nyasaland Government Officers had, during 1907 and 1908, carried out careful investigations to find :—

(*a*) Whether *G. palpalis* existed within the borders of Nyasaland? None was found, but *G. morsitans* in abundance, also *G. pallidipes, G. brevipalpis*, and *G. fusca.*

(*b*) Whether any cases of sleeping sickness were to be found in any tsetse-fly area within the Protectorate or in neighbouring districts? Over 60,000 natives were examined for enlargement of glands, and the blood of all sick natives or those with enlarged glands was examined microscopically for trypanosomes, but not a single case of sleeping sickness was found before October, 1908.

For many years previous to this, the Government and Mission doctors scattered throughout Nyasaland (about thirty in all) had examined many thousands of sick natives annually, both clinically and with the microscope, and not a single case of sleeping sickness had been encountered. Had the disease been endemic in the country, I consider it impossible that one with such outstanding clinical and microscopical features could have been missed by all the medical observers in Nyasaland.

Personally, the writer had for nine years previous to 1910 examined not only many hundreds of sick natives annually from the very forest where the disease subsequently claimed so many victims, but had

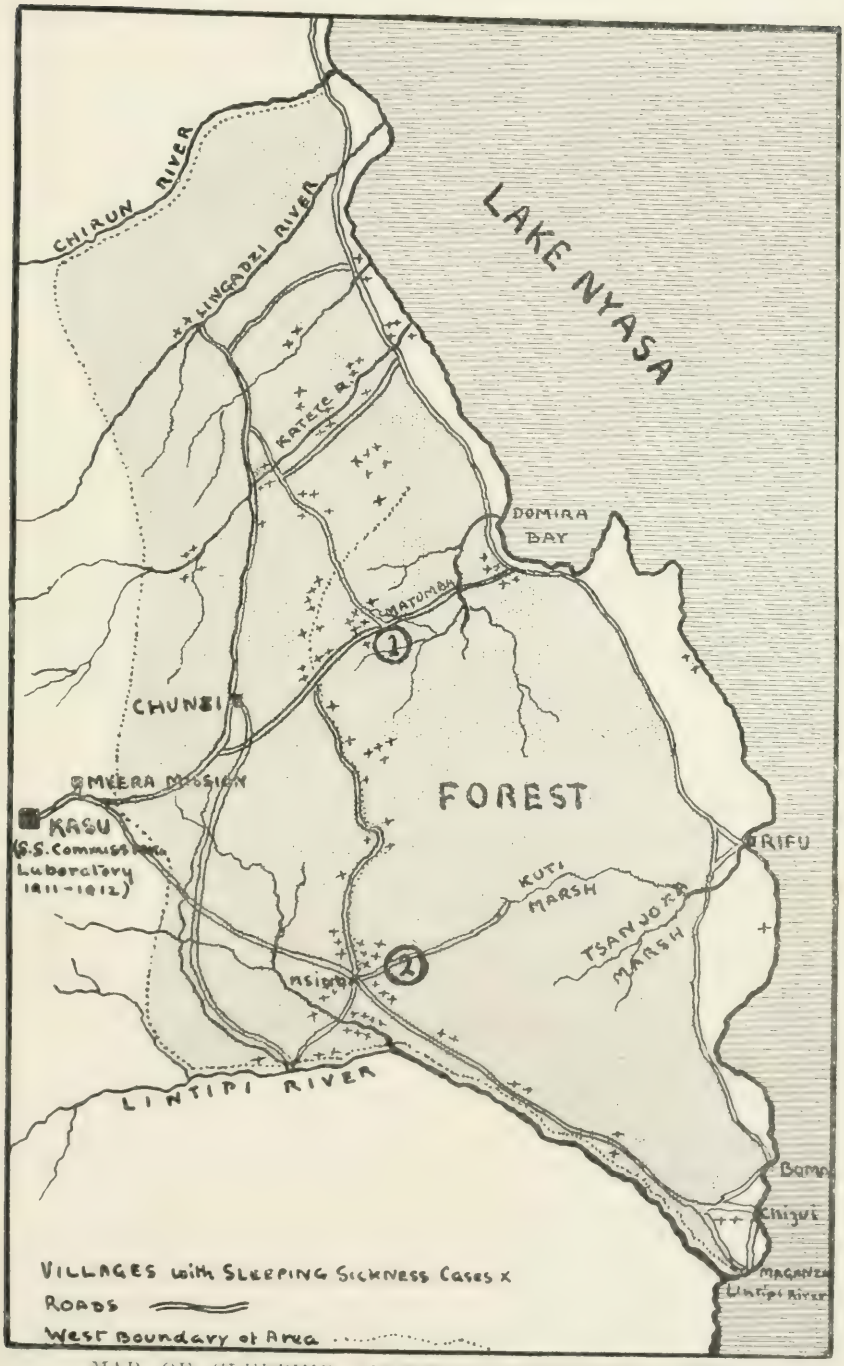
observed the health of thousands of native carriers, porters and other labourers regularly employed at Mvera Station, who were constantly traversing the portion of the neighbouring forest afterwards so heavily infected, without once meeting a case showing the marked clinical and blood features which became so common after 1910.

From 1905 he was on the look-out for cases of sleeping sickness, having been warned by accounts in the medical press of its ravages in Uganda and elsewhere.

Case 1.—In October, 1908, the first case of sleeping sickness in Nyasaland was discovered at Nkata Bay at the north end of Lake Nyasa. This patient, a native, gave a history of having travelled the previous year from his home in North Nyasa *via* Tanganyika to the Congo, and then returning *via* Cape Town to Nyasaland. His type of infection was never definitely determined, but was probably *T. gambiense* He continued for at least three years after in excellent health.

The Government resolved to establish an isolation camp for all cases that might be found, in a healthy fly-free area, and selected Dowa-Boma, about sixteen miles west of Mvera. Case 1 was accordingly brought by steamer to Domira Bay, and then conveyed in a hammock (unscreened) through twenty-five miles of forest swarming with tsetse-fly (*G. morsitans*) to Mvera, and thence to Dowa. He was accompanied by Dr. H., who was in charge of the new isolation camp, and by an armed guard. I subsequently questioned these Askari guards, who assured me that they were freely bitten by fly during the seven or eight hours they were passing through the forest. It may, therefore, safely be assumed that the patient was also bitten, and that there and then the first infection of the fly along that road occurred.

Case 2.—The second case resulted from the unfortunate accidental self-infection of Dr. H. from his patient at Dowa. As the writer visited Dr. H. and his patient at that time (November, 1908), he has no doubt whatever on this point. Dr. H. suffered from fever for some weeks, but considered it to be malarial, then improved, and undertook a visit to the forest along the Mvera-Maganga road to search for other species of tsetse-flies than *morsitans*. After about a week's sojourn in the forest he found himself too ill to work and returned to Dowa. In passing Mvera he complained to the writer that he had been badly bitten by tsetse-fly (*G. morsitans*) all the time he was in the forest. On his return to Dowa,



MAP OF SLEEPING SICKNESS PROCLAIMED AREA.

Shaded portion = Forest.

(1) Where first two cases are supposed to have infected fly.

(2) Where Case 2 (Dr. H.) was ill and infected more fly.

trypanosomes were diagnosed in his blood. He died soon after.

To judge from the rapidity of his illness his infection resembled the *rhodesiense* type rather than the *gambiense*, but the case of Capt. TULLOCK who was also accidentally infected with *T. gambiense* in Uganda, and whose illness also ran a very virulent course, seems to show that a direct infection of the *gambiense* trypanosome into a susceptible human being will enhance its virulence.

The conclusions arrived at by Dr. DUKE in Uganda (*Parasitology*, October, 1919), as to the variation in virulence of trypanosomes after direct, and after indirect infection, would seem to confirm this.

Case 3.—The third case, a native, was found at Kota Kota in May, 1909, and gave a history of having some time previously returned from the Luapula River in North-East Rhodesia, a known *T. gambiense* infested area. This patient was also taken to Domira Bay by water, and then conveyed in a similar manner to Case 1 through the forest to Mvera and Dowa. It may again safely be assumed that more tsetse-flies were infected on this occasion along that road, and that the infection was spread by means of game, which is fairly plentiful in that neighbourhood, and by natives living in that vicinity. It is noteworthy that the first locally infected cases, and most of the subsequently discovered cases, came from these two original foci, viz., villages near the Domira Bay-Mvera roads, and along the Mvera-Maganga road, where Dr. H. had stayed for some time during his illness.

Cases 4 and 5.—Cases 4 and 5 (native) were discovered at Blantyre, and the locality of their infection could not be determined, as information as to their previous movements was too vague.

Cases 6, 7, 8 and 10.—These four natives were infected during 1909 in North-East Rhodesia, at or near Luangwa Valley, the greater part of which was soon after found heavily infected by *T. rhodesiense*.

Case 9.—Was infected about December, 1909, while walking along the Nyasa Lake shore, through the forest between Domira Bay and the Maganga road. He was only discovered six months later at Zomba to have sleeping sickness.

Case 11.—Was the first case found locally infected in that forest. He lived near the Mvera-Domira Bay road, and had never been out of that district. He was brought to my hospital for treatment, and was at once recognised as suffering from a disease differing from any previously

seen in the district, but resembling the sleeping sickness cases then at Dowa.

Case 12.—Was a European, who had arrived a few weeks before from Cape Colony, and who received his infection in August, 1910, while hunting for a few days in the vicinity where Dr. H. had been ill nearly two years before.

The Government now detailed two medical officers to examine the natives of this portion of the forest thoroughly, and some thirty further cases were discovered in villages within a 20-mile radius of the centre of the Domira Bay road, most of them living near the two centres first infected. The infected area of forest bounded on the north by the Chirua River, west by the edge of the forest, south by the Lintipi River, and east by the lake shore, was proclaimed a quarantine area, and all movements of natives to and from this area were stopped.

The inhabitants of contiguous fly districts to the north and south of this area were carefully examined for cases of sleeping sickness, but not one was found until several months later a few cases of recent infection were found near Monkey Bay, about twenty miles south of the quarantine area. Other tsetse areas beyond those in the North and South Nyasa districts were, after careful medical examination of their inhabitants, declared still free of the disease in June, 1912. Soon after this date, one case occurred in the Kota Kota, and three cases in the Upper Shiré districts. Gradually the disease was found to spread more and more widely, until by 1915 practically the whole of the *morsitans* infected area of Nyasaland (stretching in a narrow belt along the west shore of the lake from Chintechi in the north, to the Murchison Cataracts on the Shiré River in the south) was found to contain infected fly, game and natives, but the degree of infection nowhere seemed to become as intense as in the part first infected near Domira Bay. Between October, 1908, and December, 1915, about 250 cases of sleeping sickness were discovered in Nyasaland. Of these only three survived for one or two years after infection, and only one lived for three years or over.

During the war all sleeping sickness investigation work in Nyasaland was stopped, and little information is available as to the subsequent progress of the disease. As far as can be gathered, however, it would seem that the pathogenicity and spread of the epidemic have both diminished, and that few cases of infection are now to be found.

A similar course of events has taken place in the Luangwa Valley in North-East Rhodesia, which was also found heavily infected in and after 1909, but where the disease has now died down to very small numbers annually.

It would seem, therefore, as if this strain (*T. rhodesiense*) in Nyasaland and Rhodesia was most virulent and spread most rapidly after its first introduction (about 1908 to 1910) into these countries, but that after many passages through the three hosts concerned (fly, game, and man), the infectivity or virulence or both has been much reduced. No opinion, however, can be hazarded as to the future until a thorough medical survey of the exact present prevalence of the disease in Nyasaland has been made.

It is not easy to explain the mode of introduction of *T. rhodesiense* into Nyasaland. According to the writer's hypothesis it would seem necessary to assume that the original source was *T. gambiense* from the north, and that by some transition at present not understood, and which has not yet been seriously investigated, the change of vector from *palpalis* to *morsitans* fly has resulted (probably after several passages through fly and game) in a different strain of trypanosome, which causes a more virulent type of disease, and can by other methods also be distinguished from the original strain.

In conclusion, the writer wishes to state that he thoroughly concurs in the conclusions arrived at by Sir David Bruce and his collaborators in Nyasaland as to the highly important rôle played by the larger varieties of game in the dissemination of the disease in Nyasaland, and in acting as a permanent reservoir of all the trypanosomes pathogenic to man and domestic animals.

A NOTE ON THE TREATMENT OF SUPPRESSION IN BLACKWATER FEVER.

BY

A. F. WALLACE, M.D., *Medical Officer, Northern Rhodesia Administration.*

A European, W. J. C., was admitted to the Broken Hill Hospital, Northern Rhodesia, on May 29th, 1921, suffering from blackwater fever. He had had blackwater fever twice before—once on the Gold Coast in 1912, and again in German East Africa in 1913. During the war he had been an officer in the Flying Corps, and he returned to Rhodesia in 1920.

Since his return he had had one severe attack of malaria in 1920, and for the six weeks prior to his admission to hospital he had repeated attacks of malaria. As was to be expected, he was not a man who took his quinine regularly.

Blackwater fever developed on the evening of May 28th. I saw him next forenoon. He was removed to hospital the same afternoon by motor car, a distance of four miles. In hospital he was given the usual routine treatment: ten grains of sodium bicarbonate every two hours and copious drinks of plain boiled water. His temperature on admission was 103°. It was subnormal next morning, and during his stay in hospital it never again rose above normal. A blood slide taken the day that he was admitted showed no malarial parasites.

The following table shows the amount of urine passed daily :—

May 29th	(10 hours)	4	ozs.	June 5th	(ten hours	5	ozs.
,, 30th	,,	10½	,,	,, 6th	,,	15	,,
,, 31st	,,	1	,,	,, 7th	,,	24	,,
June 1st	,,	0	,,	,, 8th	,,	61	,,
,, 2nd	,,	⅞	,,	,, 9th	,,	89	,,
,, 3rd	,,	2	,,	,, 10th	,,	146	,,
,, 4th	,,	3¼	,,	,, 11th	,,	141	,,

It will be seen that during the three days from May 31st to June 2nd

the patient had only passed $1\frac{7}{8}$ ozs. of urine. He had constant bilious vomiting, and was unable to retain anything by the mouth. Soon after admission, rectal salines were started, but after two days the patient gave up trying to retain them. On May 30th intravenous salines were commenced, and up to June 2nd over seven pints of saline had been injected. On June 3rd his condition seemed hopeless. Nothing could be given by the mouth or rectum, and there was marked œdema of the face, chest and arms. Hot-air baths, combined with injections of pilocarpine had been tried, and he had also been given injections of pituitrin.

At this critical period I found the following remark on the treatment of anuria in Osler's *Practice of Medicine:* "Large hot irrigations with normal salt solution with Kemp's double-current rectal tubes should be tried, as they seem to stimulate the activity of the kidneys in a remarkable way."

Acting on this, two thin œsophageal tubes (size 20) were stitched together, side by side, and with the tip of one three inches lower than the tip of the other. This was then passed per rectum high up in the bowel. At first every hour, and later every four hours, four pints of as hot saline as the patient could bear were run in from a douche can. Shortly after each irrigation the patient had a desire to micturate, and it seemed as if the irrigations had a direct effect on the blocked kidneys. The urine was at first passed in teaspoonful amounts, but it rapidly increased until, on June 10th, he passed 141 ozs. The change in his appearance from a bloated puffy individual to a thin and washed-out one was very striking.

This rectal irrigation treatment does not appear to be mentioned in books on tropical medicine. It certainly appears a rational means of applying heat to the kidneys, and must be more effectual than the application of poultices and stupes to the loins.

It would have been more dramatic if I had commenced the irrigations on June 1st, when the patient was passing no urine at all, but I am convinced that this method should be given a trial in any case of suppression.

I have to thank Dr. Aylmer May, Principal Medical Officer, Northern Rhodesia, for permission to publish this note.

A PRELIMINARY NOTE ON THE DEVELOPMENT OF *LOA LOA* (GUYOT) IN *CHRYSOPS SILACEA* (AUSTEN).

BY

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AND

S. L. M. CONNAL, M.B.E., M.A., B.Sc. (*From the Medical Research Institute, Lagos*).

We spent two months of the present year (1921), from 28th May until 20th July, in Sapele, investigating the possibility of Chrysops being the carrier of *Loa loa*.

Sir Patrick Manson's suggestion, made in 1895, and Professor Leiper's work, done in 1913, as well as our own preliminary efforts in 1917, all pointed to the likelihood of this insect being the intermediate host of the filaria.

Sapele is a trading centre on the river Benin, two days by launch east of Lagos, through the creeks. Our operations were confined to the opposite side of the river from Sapele town, were Messrs. Miller Brothers, Ltd. (of Liverpool), have a large rubber estate. Bounded by swamps as it is, and with a large labouring population, it formed an ideal site for our purpose, particularly as Mr. J. B. Methven, the manager, took a warm interest in our work, besides providing us with a bungalow and freely offering every facility. Chrysops were extremely numerous, and the percentage of labourers harbouring *Loa loa* was high. We conducted twenty feeding experiments, in which we fed six hundred and eight flies. *Chrysops silacea* predominated, and the remainder were *C. dimidiata*. Five native boys were selected as a source of the infecting feed. Three of these were chosen because the number of embryos in their blood was high, and the other two because the number was small. In all five cases, as far as one can judge by repeated (hourly) examinations of the blood on different days, the infection was a pure one.

Most of the flies used in the experiments have been preserved for section. These, except in a few instances, have not yet been examined.

One hundred and fifty-three have been dissected and each one was found to be successfully infected. The findings have been so consistent and so similar in the different series of experiments, that we feel justified in making the following preliminary statements, particularly as some considerable time must elapse before we can have all our preserved flies sectioned and examined.

Within six hours of the infecting feed, the great majority of the embryos have cast their sheath and migrated through the stomach wall to reach the abdominal muscles, where most of them come to rest. A number proceed to the muscles of the thorax and of the head. Within twenty-four hours, in a heavy infection, the young filariæ may be found in all these situations.

The embryos do not develop with equal rapidity. In some of the experiments, examinations were made at six-hourly intervals up to seventy-two hours after the infecting feed, and, in diminishing numbers as the time advanced, up to forty-eight hours, embryos which had not undergone ecdysis were found alive and active. A few die in the stomach without having cast their sheath, and a small number succumb after ecdysis. The first change, after the sheath is shed, is a shortening and broadening. Growth is rapid, but up to the third or fourth day the shape is cigar- or torpedo-like. By the fifth and sixth days, the young filariæ may be seen, considerably elongated, lying coiled up in the muscles, giving a striking appearance of whorls. All the forms are not of equal length on any given day. This difference in size is attributable, for the most part, to the difference in the time when the earliest forms migrated through the stomach wall, some coming to rest in the muscles at least forty-eight hours before others.

The preference for the abdominal muscles and the grouping in whorls are very outstanding features.

We have not observed any stages where motion entirely ceases, as is described for *Filaria bancrofti*.

The final stage of development is reached in from ten to twelve days. At this time the individuals in the abdomen and the thorax begin to travel towards the head. Some may be found in the roots of the wings and in the coxæ. They have been found even as far down as the metatarsus. It seems likely that this was due to overcrowding. When ready for another host, the fully metamorphosed forms are accumulated

at the root of the proboscis and in the labium. They gain exit by the labella.

In attempts to infect various animals (five guinea-pigs, two rabbits and a monkey), it was noted that, in a heavy infection, the fly, whilst feeding, shows signs of irritation, withdrawing and reinserting the proboscis several times. Finally, it jerks out the organ and rapidly "milks" the proboscis with the fore-legs, flicking the minute masses of filariæ some little distance away, and distributing others by moving its position. In slight infections it is difficult to detect the issue of the worms, a fleeting impression of a glistening movement being sometimes obtained. When the filariæ have reached the skin, they are just visible to the naked eye. Their movements are extremely rapid and in less than sixty seconds they have completely disappeared beneath the epidermis.

We are not able to state how long a fly may remain infective, as none of our specimens survived the infecting feed for a longer period than fourteen days. It is interesting to note, however, that of three flies found during their life-time to be doubly infected, none lived longer than one day after filariæ were observed issuing from their proboscis.

Further description is reserved until all our material has been studied.

The flies used in the experiments were caught wild in the bush. Only 30 to 40 per cent. of the individuals in one batch would feed on the day of capture. Only those flies whose stomachs were empty were allowed to feed. This precaution excluded the possibility of a natural infection contracted less than two days before, as even a small feed of blood can be readily detected through the abdominal wall, and it takes at least forty-eight hours to disappear. The remaining 60 to 70 per cent. were set aside, and some were used a day or two later for another feeding experiment. Most of them, however, were dissected on the same day or were preserved for future dissection. Still others were fed on a clean guinea-pig or on a non-infected native boy, and dissected at definite intervals. By these means we established some manner of control. The total number of controls examined so far is two hundred and twenty-five, and the number found naturally infected is eight, which is just over 3·5 per cent. In the one hundred and fifty-three experimentally-infected flies examined, five showed a double infection, that is, just over three per cent. had a naturally acquired infection before being again infected by us.

As regards the bionomics of Chrysops, we were successful in finding the eggs and the larvæ but not the pupæ. The eggs were found laid in a batch on a moist dead leaf lying on the surface of the mud of the swamp. Larvæ in different stages of development could always be found, but never close together, in the mud of the swamp, at a depth of a few inches. Very young larvæ were also found in the moisture between the leaves of the "screw-pine" and in the flower of a shrub growing on the river banks. As none of our specimens has yet pupated, we are not able to state which species prefers the mud of the swamp, or if there is any preference. We were able to induce only one captive fly to lay eggs.

C. silacea in Sapele far outnumbers the other species. *C. dimidiata* is fairly common. In nature it is very distinct from *C. silacea* in size, in colour, and in markings, and is differentiated even by the native *C. longicornis*, in our experience, is rare.

No other species of Chrysops were obtained. As regards the habits of the flies they are most troublesome in the late morning and in the early evening. They are practically noiseless, very persistent, and it is usually the act of withdrawing, not of inserting, the proboscis that is felt. They are voracious blood-suckers and seldom leave off until enormously distended.

Note by Editor.—KLEINE'S reference to the development of *Filaria loa* in Chrysops appears to have been overlooked by these observers. The exact reference and review in the *Tropical Diseases Bulletin*, 1916, vol. 7, p. 361, is as follows :—

KLEINE, (F. K.). **Die Uebertragung von Filarien durch Chrysops** (The Transmission of Filaria by Chrysops). Zeitchr. f. Hyg. u. Infektionskr. 1915. Oct. 26. Vol. 80. No. 3. pp. 345-349.

This is an account of an investigation, carried out at Eseka in the Cameroons, regarding the transmission of Filaria by Chrysops. The inhabitants of the district in and around that in which the work was carried out are heavily infected with *Filaria loa*, and Chrysops and sand-flies abound.

Six hundred female flies were examined, and, of these, five hundred were *C. dimidiata* and the remainder *C. silacea*. Of the whole number, thirty-two were found to be infected, *i.e.*, 5 per cent., and in the case of nine of these infected flies the larvæ were fully mature.

Development, according to the author's account, appears to take place in the fatty connective tissue surrounding the tracheæ in the abdominal cavity, and it is to that site that the embryos converge when they first perforate the gut of their host. In the early stages the larvæ remain motionless, and it is not until their evolution has made marked advance that they commence to leave their position in the fatty connective tissue and to wander about the abdominal cavity. Later, they make their way to the cephalic end of the body, and, in two cases, the author was able to demonstrate the emergence of the larvæ from the proboscis. This was carried out by placing the head, after removal, in a few drops of salt solution and observing under a low power of the microscope.

The size of the mature larva is given as being the length of the diameter of the field, when ocular 2 and objective AA are used, with the tube drawn out to 160mm.

[The Chrysops used in this work were, apparently, collected in a haphazard fashion, and the hosts upon which they had fed were consequently unknown. The nature of the larvæ, therefore, would appear to be undetermined. Neither illustrations nor a description of the larvæ are given, and no attempt was made to communicate filarial infection by means of infected flies. The work must be regarded as incomplete.] R. P. C.

EXTRACTS FROM THE TROPICAL DISEASES BULLETIN.

PEREZ GROVAS (P.). **Experimental Transmission of Yellow Fever Cases occurring in Vera Cruz in 1920, together with the Isolation of** *Leptospira icteroides*.—*Jl. Amer. Med. Assoc.* 1921. Feb. 5. Vol. 76. No. 6. pp. 362–365.

The chief importance of this paper lies in the fact that it gives the first confirmation of NOGUCHI'S work on *Leptospira icteroides* of yellow fever. On the occasion of an outbreak of the disease in Vera Cruz in July, 1920, after a complete immunity for many years, the writer took the opportunity of repeating some of NOGUCHI'S experiments. The result has been that guinea-pigs have been successfully inoculated with the virus of yellow fever, while the characteristic lesions in these animals and the leptospira itself, as described by NOGUCHI, have been found. The strain was easily passed from one guinea-pig to another, and cultures of the organism were also obtained. These cultures were proved to be virulent to guinea-pigs.

The paper is in the nature of a preliminary report, and a more detailed account of the investigations, which are being extended, is promised. C. M. W.

i. LANFRANCHI (A.). **Sul possible passaggio dei tripanosomi nel latte.** [On the Transmissibility of Trypanosomes in the Milk.] *Atti. R. Accad. Lincei. Rendiconti.* 1916. (5 ser.) Vol. 25. (1 sem.) p. 369.

ii. LANFRANCHI (A.). *Idem.* [2nd note.] *Ibid.* 1918. Vol. 27. (1 sem.) p. 62.

In these two communications the author records the results of some interesting experiments undertaken to ascertain whether trypanosomes can be transmitted from an infected mother to her offspring through the milk.

[The first has already been briefly noticed in this *Bulletin*, Vol 8, p. 275.]

i. The first experiments were made with dogs, using four different species of trypanosome. The method employed was as follows: A bitch was inoculated, immediately after parturition, with 2 c.c. of the citrated blood of an infected guinea-pig. Her milk was afterwards tested, at intervals, both by direct microscopic examination and by intraperitoneal inoculation into rats and mice, in order to ascertain whether it contained trypanosomes. Her pups were also examined, by the same methods, to discover whether they had contracted her infection. The strains of trypanosomes used were obtained from the Institut Pasteur.

1. *T. brucei.* In the milk of the bitch inoculated with this organism no trypanosomes were ever discoverable microscopically, but one of the rats into which it was injected became infected, and died of trypanosomiasis. All the four pups of this bitch died, and were found to have acquired their mother's infection; for although the blood of all gave negative results when examined under the microscope, some of the rats inoculated from each pup became infected with *T. brucei*.

2. *T. rhodesiense.* All the pups belonging to the bitch infected with this trypanosome remained healthy. No trypanosomes were demonstrable in them microscopically, and none of the mice into which their blood was injected acquired infection. The milk of the mother was always negative microscopically; but on one occasion a mouse into which it was injected gave a positive result—acquiring a fatal infection with *T. rhodesiense*.

3. *T. evansi.* The milk of the bitch inoculated with this trypanosome remained completely negative to all tests. Her pups also all gave wholly negative results—in trypanosomes being discoverable in their blood microscopically, or by animal inoculation.

4. *T. gambiense.* Of the seven pups belonging to the bitch inoculated with this trypanosome, six gave no evidence of infection; but one acquired its mother's infection, for injection of its blood into a mouse gave a positive result. The milk of the mother was

microscopically negative throughout, but on one occasion its injection into a mouse was followed by a positive result—the mouse succumbing to a *T. gambiense* infection.

From the foregoing experiments the author concludes that *T. brucei*, *T. rhodesiense*, and *T. gambiense* can pass into the milk of an infected bitch : and further that, in the case of *T. brucei* and *T. gambiense*, the mother can transmit the trypanosomes to her offspring in the process of suckling them.

ii. The second paper describes further experiments made by the same methods but using different animals for inoculation. The same four species of trypanosome were employed, and in addition the strain which the author calls "*T. lanfranchii*"—the organism with which he himself accidentally became infected, [See this *Bulletin*, Vol. 8, p. 274.]

1. *T. brucei*. Three female rats and one guinea-pig almost entirely negative results after inoculation with this trypanosome. All their offspring remained uninfected. The milk of the mothers gave negative results on all occasions save one, when one of the mice injected with the milk of one rat proved positive.

2. *T. rhodesiense*. Two rats and a guinea-pig were inoculated (the latter before parturition) with this trypanosome. Microscopic examination and inoculation tests carried out with the milk of the mothers and the blood of the offspring all gave completely negative results.

3. *T. eransi*. Two female rats and a guinea-pig inoculated with this species again gave results which were chiefly negative. All the offspring remained uninfected. The milk of all the mothers appeared negative to microscopic examination ; but its injection into other animals gave positive results on two occasions—samples of the milk of one rat and of the guinea-pig each once causing infection of mice into which they were injected.

4. *T. gambiense*. Similar experiments carried out with this trypanosome, injected into two rats and a guinea-pig, gave negative results throughout. No evidence was obtained indicating that the trypanosomes had passed into the milk or the young.

5. "*T. lanfranchii*." Two strains, isolated at different times from the author's blood, were employed. Two rats and two guinea pigs were inoculated. All tests of the milk and the offspring were negative with two exceptions. On one occasion the milk of one of the mother rats gave a positive result when injected into mice ; and one of the young guinea-pigs died, its blood, when injected into rats, proving positive. All the other young apparently remained uninfected.

A cat, which gave birth to a single kitten, was also inoculated with this strain. No trypanosomes could be detected afterwards in the cat's milk or in the kitten, either by microscopic examination or by animal inoculation.

The same trypanosome was also injected into a mare, in milk at the time, but without a foal. Microscopic examination of her milk subsequently revealed the presence of scanty trypanosomes in it on several occasions. Inoculation of the milk into rats also gave positive results.

From this series of experiments the author concludes that *T. brucei*, *T. eransi*, and "*T. lanfranchii*," are able to pass into the milk of an infected mother rat ; and that *T. eransi* and "*T. lanfranchii*" can pass into the milk of the guinea-pig, as well as into that of the rat. "*T. lanfranchii*," moreover, can pass not only into the mother guinea-pig's milk, but also by this channel into the young which she suckles. Finally, this trypanosome can be demonstrated in the milk of an experimentally infected mare. The author recalls that NATTAN-LARRIER (*Rev. de Path. Comp.*, 1913, Dec., p. 282) had previously found that *Schizotrypanum cruzi* constantly passes into the milk, and *T. equiperdum* does so occasionally. He also refers to the observations of VELU and EYRAUD (1916), who state that a bitch infected with the horse trypanosome of Morocco communicated the infection to one of her pups in the process of lactation. [See this *Bulletin*, vol. 9, p. 34.]

[The foregoing experiments appear to prove, therefore, that trypanosomes are able to infect young animals by way of their mother's milk. They can be demonstrated sometimes in the milk of an infected mother—by direct microscopic examination, or by inoculation of the milk into susceptible animals.]

The large proportion of positive results will be noted. It has been demonstrated, apparently, that—at least in certain hosts—*T. brucei*, *T. rhodesiense*, *T. gambiense*, "*T. lanfranchii*," and *T. evansi*, are all able to get into the milk of an infected animal, and there remaining in an infective condition. Infection on the offspring by imbibing the infected milk has been demonstrated in the case of *T. brucei*, *T. gambiense*, and "*T. lanfranchii*." It is conceivable that the milk was not the real route of infection of the young animals in these experiments. LANFRANCHI discusses other possibilities, but believes that his method of experimentation excluded them, or rendered an alternative interpretation highly improbable. It seems worthy of note that the trypanosomes studied were all pathogenic species in susceptible animals which cannot be regarded as their normal hosts. C. D.

MCDONALD (J. T.). **Treatment of Leprosy with the Dean Derivatives of Chaulmoogra Oil; Apparent Cure in 78 Cases.**—*Jl. Amer. Med. Assoc.* 1920. Nov. 27. Vol. 75. No. 22. pp. 1,483-1,487.

The Leprosy Investigation Station of the United States Public Health Service, established in Honolulu in 1905 and on Molokai, Hawaii, in 1906, and of which Dr. J. T. McDonald is the Director, has made many important contributions to the study of leprosy. Since 1918, the station has had the help of Dr. A. L. DEAN who following up the work of POWER on the chaulmoogric fatty acid series, has overcome the difficulty presented by the insolubility of these fractions of chaulmoogra oil by converting them into their ethyl esters, thus obtaining "thinly fluid oils readily absorbable from intramuscular injection." The fact that the Director of the Investigation Station is also Superintending Physician to the Kalihi Hospital has brought about a very satisfactory co-operation between the laboratory and the wards, and has greatly facilitated the work of research on these products. The standard preparation now used for weekly injection consists of the ethyl esters of the entire fatty acids of the whole oil with 2 per cent. of iodine by weight, chemically combined. The dosage begins with 1 c.c. as in intramuscular injection and is increased by 1 c.c. at every second or third injection, until from 2 to 6 c.c. is reached, according to the age and weight of the patient. For internal use, the mixed fatty acids, carrying 2·5 per cent. of iodine in chemical combination, are given in capsules, as it is thought that the fatty acids themselves are more likely to conform to the normal digestive processes than their ethyl esters. "We are therefore using by mouth a predigested oil or fat which is semi-solid at room temperature, and, in capsule, very easy to take. Its dosage begins with $\frac{1}{4}$ gm. per 100 lbs. of the patient's weight, three times a day, an hour or two after meals. This is gradually increased every two weeks until the maximum of 1 gm. per 100 lbs. of weight per dose is reached." Preparatory to injections, syringes, needles, etc., are sterilized for upwards of 30 minutes in the autoclave. An "all-glass" 20 c.c. Paris syringe with an antitoxin needle is used, the needle being inserted its full length into the muscles at a site in the upper and outer quarter of the gluteal region, after treatment of the skin by iodine. The author has only noted one abscess in a series of close on 7,000 injections by this method.

Some interesting references are made to leprosy fever, a phenomenon which appears to arise *pari passu* in patients when under treatment, and which, while decidedly disagreeable, is found to subside in a short time and to be followed by decided amelioration in the patient's condition. The temperature rises and, within a few days, there appear on the face, forearms, or elsewhere, "a number of well-defined, circumscribed eruptions, usually circular, dull red, and raised from the level of the surrounding skin, thickest in the centre, and hot and evidently more or less tender to the touch." These elevations are not leprosy nodules, hardly ever contain bacilli, and tend to recede and disappear. Speaking of the diagnosis of leprosy, the author states that he has abandoned the opinion that "the microscope is the supreme agent of the final diagnosis of leprosy." He now considers that leprosy can be diagnosed clinically in some cases at times when the bacilli cannot be found, especially in nerve cases. Since October 1, 1918, no less than 78 patients have been discharged "on parole," apparently cured. These comprised 36 males and 42 females; the average age on admission was $23\frac{1}{2}$ years, 49 were of the nodular type, 24 anæsthetic and 5 mixed. Of the total number, 55 were bacteriologically positive on admission and 23 were negative. S. L. C.

i. PRICE (J. Dodds). **Antimony in Kala Azar.** *Brit. Med. Jl.* 1920. Sept. 18. pp. 453–454.

ii. ELWES (F. F.). **Antimony in Kala Azar.**—*Ibid.* 1920. Oct. 16. p. 610.

i. The writer of this letter draws attention to the superiority of the sodium salt of antimony tartarate over the potassium salt, which is much more toxic. The former is just as efficacious as the latter in its specific action in kala azar. He has personally given more than 3,000 intravenous injections of the drug and has watched the effect of over twice that number given by his assistants in Assam. The conclusion arrived at is that in this salt we possess the means of curing the vast majority of cases of kala azar, provided they are secured in the early stages of the disease. A young tea-planter who first showed the disease in September was perfectly fit by Christmas after having received 21 injections, representing 25 grains of the sodium salt. He was free from fever after the third injection. Beyond an occasional purge no other treatment was given. Another case, which did not commence treatment so early, was treated in India, but left for England before the treatment was complete. Though his fever had ceased and he had put on two stone in weight on the voyage home, he relapsed in England, where he was further treated by the writer. After the seventh injection his fever left him and he has remained free since. Between July 28 and August 12 injections were given every other day. The initial dose was 4 c.c., and this was increased by 1 c.c. at each injection, the strength of the solution being $1\frac{1}{2}$ gr. in 10 c.c. of normal saline. The patient has now had 13 injections and is still under treatment. The object of referring to these two cases is to point out that with the sodium salt the fever in kala azar, even when the case is not of recent origin, is often lost quite as quickly as in the case successfully treated with acetyl-*p*-aminophenyl stibiate of soda referred to by Dr. MANSON-BAHR in the *Brit. Med. Jl.* of August 14, [see this *Bulletin*, Vol. 17.]

The writer prepares his own solutions of the drug, using normal saline and sterilising by placing the bottle in boiling water in a pan for 10 minutes. The solution can be repeatedly boiled without destroying its efficiency. Injections are given when the patient's stomach is empty, and he is made to lie down for half an hour afterwards. If there is the slightest tendency to cough after the injection the next dose is reduced by $\frac{1}{2}$ cc. With the sodium salt, however, this is rarely necessary. The majority of the cases treated by the writer in Assam were cured after 30 gr. of the drug. A cure was considered as having taken place provided fever had been absent and the spleen markedly diminished in size and if no febrile reaction occurred after the injection of the drug. If parasites are still present there is usually a rise, sometimes only a slight one, after an injection, and it is an indication for further treatment.

ii The letter by ELWES contains some comments on the one just reviewed. The writer says that he has used a 2 per cent. solution of tartar emetic for kala azar and has carried the dose to 10 c.c. of the solution. When the dose has attained 6 c.c., severe cough, nausea and retching occur. By giving the dose on an empty stomach and keeping the patient absolutely still for 10 minutes after injection these symptoms are minimized. The writer thinks the use of the sodium salt as employed to such a large extent by DODDS PRICE is a distinct advance, but considers it possible that if a 2 per cent. solution were used instead of the $1\frac{1}{2}$ grains in 10 c.c. as advised by that writer, unpleasant symptoms might also appear. However, the sodium salt seems to have a greater curative value than the potassium one, and hence can be used in smaller doses. He wishes to endorse the view that a cure cannot be said to have taken place unless there has been marked diminution in the size of the spleen. That sign he considers of the greatest importance. If the patient is discharged with only a slight reduction in the spleen, then relapse occurs, and subsequent treatment is very unpromising, whereas if the spleen has been considerably reduced it will continue to decrease in size after treatment has been suspended. When once treatment has been commenced it is most important to continue the injections until the apparent cure has been attained. C. M. W.

CORRESPONDENCE.

SLEEPING SICKNESS.

The following extract from a letter, dated 7th May, 1921, from Dr. George Prentice, of Kasunga, N'gara, Nyasaland, may be of interest to Fellows :—

"You may be interested to know that with the cessation of free shooting in this area there is a very rapid advance of tsetse, and herds of cattle are going down before it, kraals being cleared; also that sleeping sickness has extended from the area where Dr. Yorke was working in Rhodesia, and has now crossed the Nyasaland border north of Fort Jamieson. My first case from this side of the border was probably a carried one, but now the disease seems to have got a hold in two villages.

"Four members of one family died and a child in another. The mother of that child is also affected, and is at present a patient here.

"If any of your maps show Mngwazu's village, on the Lingadzi, about 13° south, that is the principal village.

"These cases are occurring at an altitude of just 3,500 feet."

LIST OF FELLOWS.

The following have been duly elected Fellows of the Society:—

Nathaniel Crichlow, M.B., Ch.B. (Glas. Univ.), British Solomon Islands.

Henry Eustace Cross, A.Sc. (Dur.), M.R.C.V.S., D.V.H. (Liv.), Capt. R.A.V.C., India.

Harry Lovett Cumming, M.D., Ch.B. (Edin.), D.P.H., D.T.M. & H. (Camb.), Shanghai, China.

William Muir Howells, M.B., Ch.B., D.T.M., c/o Holt & Co.

Ralph W. Mendelsohn, M.D. (Chicago), Medical Officer of Health, Bangkok, Siam.

Raymond William Orpen, L.R.C.P., L.R.C.S. (Ire.), D.P.H., Lagos, West Africa.

Edward Halford Ross, L.R.C.S., (Edin.), London, W.

Robert Gordon Simpson, M.R.C.S., L.R.C.P., M.B., B.S., D.T.M., Winchmore Hill, London.

Henri Tejera, M.D., Director of the Public Health Laboratory, Venezuela.

Harry Morton Stanley Turner, D.T.M. (Lond.), Wing-Commander R.A.F.M.S., Air H.Q., Halton Camp.

MOVEMENTS OF FELLOWS.

The following Fellows have notified the Secretaries that they are at present in England:—

Major C. H. Bowle-Evans, C.M.G., India.
H. F. Conyngham, L.R.C.P., Borneo.
Major F. W. Cragg, M.D., I.M.S., India.
J. B. Davey, M.B., M.R.C.S., Tanganyika.
H. Lyndhurst Duke, B.A., B.C., M.D., Uganda.
Colonel A. F. W. King, F.R.C.S., India.
J. Scott Macfie, M.B., Gold Coast.
P. A. Maplestone, D.S.O., Australia.
Claude H. Marshall, M.R.C.S., L.R.C.P., Uganda.
F. W. O'Connor, M.R.C.S.
W. H. Peacock, M.B., M.R.C.S., Sierra Leone.
A. Pearson, M.B.
Arthur Pickels, M.B., B.S., Gold Coast.
J. Macfarlane Pollard, M.B., Nigeria.
A. T. Stanton, M.D., Malay States.
R. Y. Stones, M.C., M.D., Kenya Colony.

H. S. Hollenbeck, M.D., Angola, is at present in U.S.A.

EDITORIAL NOTICES.

The Editor of the TRANSACTIONS will be pleased to receive short papers to be included under the heading of "Notes and Comments" or "Questions," in addition to longer papers on subjects of Tropical interest, which may be offered for presentation at meetings of the Society. He will also be glad to receive books for review.

Attention is drawn to the rule that Contributors of papers to the TRANSACTIONS receive 25 reprints free of charge, *provided they apply for these when sending the paper.*

Any communication for the Editor should be addressed to THE EDITOR of the TRANSACTIONS of the Royal Society of Tropical Medicine and Hygiene, 11, Chandos Street, Cavendish Square, W. 1.

PUBLISHING NOTICES.

NOTICE TO FELLOWS.

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All communications should be addressed to—

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